L Number	Hits	Search Text	DB	Time stamp
2	2	9851331.pn. and somatostatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:34
7	2	6123916.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 06:52
8	2	6123916.pn. and somatostatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 06:52
5	2	4853371.pn. and somatostatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:07
9	0	4853371.pn. and somatostatin and obesity	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:07
10	0	4853371.pn. and somatostatin and fat	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:07
11	2	4853371.pn. and somatostatin and insulin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
12	0	4853371.pn. and somatostatin and insulin and weight	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
13	0	4853371.pn. and somatostatin and insulin and calorie	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
14	0	4853371.pn. and somatostatin and insulin and caloric	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
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20	1	somatostatin.clm. and insulin.clm. and obesity.clm. and hypersecretion	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:35
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22	7	somatostatin and insulin SAME hypersecretion and obesity	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:37

23	3	somatostatin.clm. and insulin SAME hypersecretion and obesity	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:37
24	2	somatostatin.clm. and insulin SAME hypersecretion and obesity.clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:38
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19	20	somatostatin.clm. and insulin.clm. and obesity.clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:39
26	12	obesity.ab. and somatostatin.clm. and insulin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:39
27	10	obesity.ab. and somatostatin.clm. and insulin.clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:40
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29	22	514/2.ccls. and somatostatin and obesity and insulin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:40

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$0.00 0.134 DialUnits FileHomeBase
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File 155:MEDLINE(R) 1966-2003/Sep W4
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Set	Items	Description
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	10	AU=LUSTIG, R?
	129	AU=LUSTIG R?
S1	139	AU=((LUSTIG, R?) OR (LUSTIG R?))
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	45966	SOMATOSTATIN
S2	5	S1 AND SOMATOSTATIN
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Display 2/2/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11919034 99362531 PMID: 10431109
Hypothalamic obesity caused by cranial insult in children: altered glucose and insulin dynamics and reversal by a somatostatin agonist.
Lustig R H; Rose S R; Burghen G A; Velasquez-Mieyer P; Broome D C; Smith K; Li H; Hudson M M; Heideman R L; Kun L E
Department of Pediatrics, University of Tennessee, Memphis, USA.
Journal of pediatrics (UNITED STATES) Aug 1999, 135 (2 Pt 1) p162-8, ISSN 0022-3476 Journal Code: 0375410
Contract/Grant No.: M01-RR00211; RR; NCRR
Comment in J Pediatr. 1999 Aug;135(2 Pt 1) 142-4; Comment in PMID 10431105
Document type: Clinical Trial; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

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2/2/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11919034 99362531 PMID: 10431109
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Comment in J Pediatr. 1999 Aug;135(2 Pt 1) 142-4; Comment in PMID 10431105
Document type: Clinical Trial; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: AIM; INDEX MEDICUS
Tags: Animal; Female; Human; Male; Support, U.S. Gov't, P.H.S.
Descriptors: *Brain Damage, Chronic--complications--CO; *Hormones, Synthetic--therapeutic use--TU; *Hypothalamic Diseases--drug therapy--DT; *Obesity--drug therapy--DT; *Octreotide--therapeutic use--TU; *Somatostatin --agonists--AG; Adolescent; Child; Disease Models, Animal; Hyperphagia --drug therapy--DT; Hyperphagia--etiology--ET; Hyperphagia--physiopatholo gy--PP; Hypothalamic Diseases--etiology--ET; Hypothalamic Diseases

--physiopathology--PP; Insulin--blood--BL; Obesity--etiology--ET; Obesity --physiopathology--PP; Rats
CAS Registry No.: 0 (Hormones, Synthetic); 11061-68-0 (Insulin); 51110-01-1 (Somatostatin); 83150-76-9 (Octreotide)
Record Date Created: 19990824
Record Date Completed: 19990824
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Display 2/2/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10110295 22074690 PMID: 12079271
Use of somatostatin receptor ligands in obesity and diabetic complications.
Boehm Bernhard O; Lustig Robert H
Division of Endocrinology, Ulm University, Robert-Koch-Strasse 8, Ulm/Donau, 89070, Germany.
Best practice & research. Clinical gastroenterology (England) Jun 2002, 16 (3) p493-509, ISSN 1521-6918 Journal Code: 101120605
Document type: Journal Article; Review; Review Literature
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Subfile: INDEX MEDICUS
Tags: Human
Descriptors: *Diabetic Retinopathy--drug therapy--DT; *Obesity --drug

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T S2/6/1-5

2/6/1 (Item 1 from file: 155)
11919034 99362531 PMID: 10431109
Hypothalamic obesity caused by cranial insult in children: altered glucose and insulin dynamics and reversal by a somatostatin agonist.
Aug 1999

2/6/2 (Item 2 from file: 155)
10110295 22074690 PMID: 12079271
Use of somatostatin receptor ligands in obesity and diabetic complications.
Jun 2002

2/6/3 (Item 1 from file: 73)
11958336 EMBASE No: 2003069647
Autonomic dysfunction of the beta-cell and the pathogenesis of obesity
2003

2/6/4 (Item 2 from file: 73)
11631097 EMBASE No: 2002202317
Hypothalamic obesity: The sixth cranial endocrinopathy
2002

2/6/5 (Item 3 from file: 73)
10654494 EMBASE No: 2000120584
Hypothalamic obesity caused by cranial insult in children: Altered glucose and insulin dynamics and reversal by a somatostatin agonist
1999
?
T S2/3,AB/1-5

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2/3,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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11919034 99362531 PMID: 10431109
Hypothalamic obesity caused by cranial insult in children: altered glucose and insulin dynamics and reversal by a somatostatin agonist.
Lustig R H; Rose S R; Burghen G A; Velasquez-Mieyer P; Broome D C; Smith K; Li H; Hudson M M; Heideman R L; Kun L E
Department of Pediatrics, University of Tennessee, Memphis, USA.
Journal of pediatrics (UNITED STATES) Aug 1999, 135 (2 Pt 1) p162-8, ISSN 0022-3476 Journal Code: 0375410
Contract/Grant No.: M01-RR00211; RR; NCRR
Comment in J Pediatr. 1999 Aug;135(2 Pt 1) 142-4; Comment in PMID 10431105
Document type: Clinical Trial; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
OBJECTIVE: Hypothalamic obesity is a rare sequela of cranial insult, for which pathogenesis and treatment remain obscure. In rodents ventromedial hypothalamic damage causes hyperphagia, obesity, hyperinsulinism, and insulin resistance. Reduction of insulin secretion in humans may attenuate weight gain. METHODS: Eight children with intractable obesity after therapy for leukemia or brain tumors underwent oral glucose tolerance testing (OGTT) with simultaneous insulin levels before and after treatment with octreotide for 6 months. RESULTS: In comparison with a 6-month pre-study observation period, patients exhibited weight loss (+6.0 +/- 0.7 kg vs -4.8 +/- 1.8 kg; P =.04) and decrease in body mass index (+2.1 +/- 0.3 kg/m(2) vs -2.0 +/- 0.7 kg/m(2); P =.0001). Recall calorie count decreased during the 6 months of treatment (P =. 015). OGTT demonstrated biochemical glucose intolerance in 5 of 8 patients initially and in 2 of 7 at study end, whereas insulin response was decreased (281 +/- 47 microU/mL vs 114 +/- 35 microU/mL; P =.04). Percent weight change correlated with changes in insulin response (r = 0.72, P =.012) and changes in plasma leptin r = 0.76, P =.0004). CONCLUSIONS: Patients with hypothalamic obesity demonstrate excessive insulin secretion. Octreotide administration promoted weight loss, which correlated with reduction in insulin secretion on OGTT and with reduction in leptin levels. Pre-study biochemical glucose tolerance improved in several patients while they were receiving octreotide. These results suggest that normalization of insulin secretion may be an effective therapeutic strategy in this syndrome.

2/3,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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10110295 22074690 PMID: 12079271
Use of somatostatin receptor ligands in obesity and diabetic complications.
Boehm Bernhard O; Lustig Robert H
Division of Endocrinology, Ulm University, Robert-Koch-Strasse 8, Ulm/Donau, 89070, Germany.
Best practice & research. Clinical gastroenterology (England) Jun 2002, 16 (3) p493-509, ISSN 1521-6918 Journal Code: 101120605
Document type: Journal Article; Review; Review Literature
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Somatostatin (SMS) is a potent inhibitory molecule. It inhibits both exocrine and endocrine secretory functions of the pancreas, suppresses

growth hormone secretion and reduces the level of insulin-like growth factor-1. Long-acting somatostatin analogues were currently investigated for potential clinical benefits in two settings: (a) control of hyperinsulinaemia in obesity and (b) control of an excess of pro-angiogenic factors in diabetes-associated retinal complications. In two randomized, controlled trials the long-acting somatostatin analogue octreotide retarded progression of the microvascular complications in pre-proliferative and advanced stages of diabetic retinopathy. Inhibition of the early phase of insulin secretion by use of octreotide in patients with hypothalamic obesity resulted in weight loss and improved quality of life. Efficacy of octreotide correlated to residual beta-cell activity prior to the treatment. Obesity and diabetes mellitus are the most common chronic metabolic disorders in the world. The use of somatostatin analogues addressing the various hormonal imbalances of these disorders may provide a novel concept for their pharmacological treatment. Copyright 2002 Elsevier Science Ltd.

2/3,AB/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

11958336 EMBASE No: 2003069647
Autonomic dysfunction of the beta-cell and the pathogenesis of obesity
Lustig R.H.
Prof. R.H. Lustig, Department of Clinical Pediatrics, Division of Endocrinology, University of California, San Francisco, CA 94143-0136 United States
AUTHOR EMAIL: rlustig@peds.ucsf.edu
Reviews in Endocrine and Metabolic Disorders (REV. ENDOCR. METAB. DISORD.) (Netherlands) 2003, 4/1 (23-32)
CODEN: REMDC ISSN: 1389-9155
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 76

In this review, we describe and characterize a specific subtype of obesity with organic underpinnings. There is evidence for etiology (vagal modulation of beta-cell depolarization), pathogenesis (insulin hypersecretion), diagnosis (insulin dynamics during OGTT), and treatment (insulin suppression through beta-cell somatostatin receptor agonism). Although the number of obese patients with organic VMH damage is exceedingly small, the numbers of subjects who may manifest similar pathogeneses, with either a genetic, neural, or hormonal etiology, may be much greater. Studies are now underway to determine the incidence of this disorder, and the best method for diagnosis and treatment. This recognition of this syndrome of autonomic dysfunction of beta-cell insulin secretion is an important first step in improving the nosology of obesity, tying the hypothalamus to the adipocyte, and trying to correlate biochemistry with human behavior. In doing so, it is anticipated that the clinical evaluation of obesity will take a more scientific tone in the near future.

2/3,AB/4 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

11631097 EMBASE No: 2002202317
Hypothalamic obesity: The sixth cranial endocrinopathy
Lustig R.H.
Dr. R.H. Lustig, Division of Pediatric Endocrinology, Univ. of California San Francisco, Box 0136, 500 Parnassus Avenue, San Francisco, CA 94143-0136 United States
AUTHOR EMAIL: rlustig@peds.ucsf.edu

Endocrinologist (ENDOCRINOLOGIST) (United States) 2002 12/3 (210-217)
CODEN: EDOCE ISSN: 1051-2144
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 70

The hypothalamus is "ground zero" for the neuroendocrine control of five hormonal systems, which are mediated through negative feedback regulation of pituitary hormone release. Energy balance is regulated by a more complex neuroendocrine feedback loop. The hypothalamus integrates peripheral neural and hormonal afferent signals of satiety and energy reserve and directs neuroendocrine efferent arms to effect energy storage versus expenditure; however, in this feedback loop, the pituitary is not integral. Damage to this hypothalamic control system results in a syndrome of intractable weight gain. This syndrome of hypothalamic obesity is usually caused by cranial insult, such as brain trauma, tumor, surgery, or radiation. In some cases, however, it may have a congenital cause. The cause and pathogenesis of obesity in such subjects is akin to an animal model of obesity in which the ventromedial hypothalamus (VMH) is destroyed or deafferented. The VMH-lesioned rat exhibits a vagally mediated potentiation of insulin secretion in response to glucose. Excess insulin secretion favors and promotes partitioning of energy substrate into fat, even with caloric restriction. Similarly, patients with hypothalamic obesity exhibit insulin hypersecretion. By suppressing insulin release at the beta cell in a specific manner using the somatostatin agonist octreotide, the shunting of energy substrate to adipose is attenuated. Treated patients exhibit weight loss and improved quality of life, which correlate with insulin suppression. Thus, hypothalamic obesity is the sixth cranial endocrinopathy, with an identifiable cause, pathogenesis, diagnosis, and treatment.

2/3,AB/5 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

10654494 EMBASE No: 2000120584
Hypothalamic obesity caused by cranial insult in children: Altered glucose and insulin dynamics and reversal by a somatostatin agonist
Lustig R.H.; Rose S.R.; Burghen G.A.; Velasquez-Mieyer P.; Broome D.C.; Smith K.; Li H.; Hudson M.M.; Heideman R.L.; Kun L.E.
Dr. R.H. Lustig, Department of Pediatrics, Methodist LeBonheur Child. Med. Ctr., 50 North Dunlap, Memphis, TN 38103 United States
Journal of Pediatrics (J. PEDIATR.) (United States) 1999, 135/2 I (162-168)
CODEN: JOPDA ISSN: 0022-3476
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 42

Objective: Hypothalamic obesity is a rare sequela of cranial insult, for which pathogenesis and treatment remain obscure. In rodents ventromedial hypothalamic damage causes hyperphagia, obesity, hyperinsulinism, and insulin resistance. Reduction of insulin secretion in humans may attenuate weight gain. Methods: Eight children with intractable obesity after therapy for leukemia or brain tumors underwent oral glucose tolerance testing (OGTT) with simultaneous insulin levels before and after treatment with octreotide for 6 months. Results: In comparison with a 6-month pre-study observation period, patients exhibited weight loss (+6.0 +/- 0.7 kg vs -4.8 +/- 1.8 kg; P = .04) and decrease in body mass index (+2.1 +/- 0.3 kg/msup 2 vs -2.0 +/- 0.7 kg/msup 2; P = .0001). Recall calorie count decreased during the 6 months of treatment (P = .015). OGTT demonstrated biochemical glucose intolerance in 5 of 8 patients initially and in 2 of 7 at study

end, whereas insulin response was decreased (281 +/- 47 muU/mL vs 114 +/- 35 muU/mL; P = .04). Percent weight change correlated with changes in insulin response (r = 0.72, P = .012) and changes in plasma leptin r = 0.76, P = .0004). Conclusions: Patients with hypothalamic obesity demonstrate excessive insulin secretion. Octreotide administration promoted weight loss, which correlated with reduction in insulin secretion on OGTT and with reduction in leptin levels. Pre-study biochemical glucose tolerance improved in several patients while they were receiving octreotide. These results suggest that normalization of insulin secretion may be an effective therapeutic strategy in this syndrome.
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